

U.S. Department of Labor

Office of Administrative Law Judges
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Issue date: 30Nov2001

In the Matter of:

WILLIAM H. CARSON
Claimant

V.

Case No. 2000-BLA-00236

WESTMORELAND COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

Mary Zanolli Natkin, Esq.
Stephen DeLisle
For the Claimant

Mary Rich Maloy, Esq.
For the Employer

BEFORE: EDWARD TERHUNE MILLER
Administrative Law Judge

DECISION AND ORDER - AWARDING BENEFITS

Statement of the Case

This proceeding involves a third claim for benefits under the Black Lung Benefits Act, as amended, 30 U.S.C. 901 *et seq.* (hereinafter "the Act") and regulations promulgated thereunder.¹ Since this claim

¹ All applicable regulations which are cited are included in Title 20 of the Code of Federal Regulations, unless otherwise indicated, and are cited by part or section only. Director's Exhibits are indicated as "DX", Transcript of the

was filed in 1997, Part 718 applies. Because the Claimant Miner was last employed in the coal mine industry in West Virginia, the law of the United States Court of Appeals for the Fourth Circuit controls (DX 2). *See Shupe v. Director, OWCP*, 12 B.L.R. 1-200, 1-202 (1989)(*en banc*).

The Claimant, William H. Carson, filed his first claim for benefits on July 25, 1979 (DX 44-1). The claim was denied by Administrative Law Judge John C. Holmes in a decision and order dated January 27, 1984 (DX 44-42). Judge Holmes found well in excess of ten years of coal mine employment, ending in November 1981, when the Claimant suffered a stroke², and a history of smoking one-half pack of cigarettes a day for about thirty years. He found that the x-ray evidence triggered the rebuttable presumption of total disability due to pneumoconiosis found at §727.203(a)(1), but also found that the Employer established rebuttal pursuant to §727.203(b)(3). The Benefits Review Board (Board) affirmed the denial on August 27, 1986 (DX 44-53). The United States Court of Appeals for the Fourth Circuit affirmed the Board's decision in an order dated August 25, 1987 (DX 44-59).

Claimant filed his second claim for benefits on December 29, 1988 (DX 43-1). The claim was denied by Administrative Law Judge Martin J. Dolan, Jr. on October 6, 1992 (DX 43-51). Judge Dolan accepted the Employer's stipulation of fifteen years of coal mine employment and the existence of simple pneumoconiosis arising out of coal mine employment. He found the Claimant totally disabled but concluded that the cause of the disability was not a respiratory disease but impairment to his breathing apparatus brought on by his 1981 stroke. Judge Dolan also concluded that any respiratory disability did not arise out of coal mine employment. The Board affirmed the denial in a decision issued November 22, 1994 (DX 43-61). *Carson v. Westmoreland Coal Co.*, 19 BLR 1-21 (1994). In a published decision dated August 26, 1996, the Board, pursuant to the Director's motion to reconsider its holding that loss of lung function due to extrinsic factors does not constitute respiratory or pulmonary disability, deleted the sentence, "The disabling loss of lung function due to extrinsic factors, e.g., loss of muscle function due to a stroke, does not constitute respiratory or pulmonary disability pursuant to Section 718.204(c)," from its prior decision. *Carson v. Westmoreland Coal Co.*, BRB No. 93-0459 BLA (1996).

The instant claim was filed by the Claimant on November 24, 1998 (DX 1). The Employer was given notice of the claim on December 16, 1998, (DX 19), and controverted liability on December 21, 1998 (DX 20). The District Director, Office of Workers' Compensation Programs ("OWCP") made a determination on March 16, 1999, denying benefits (DX 17). The Claimant submitted additional evidence, and the District Director made an initial finding of entitlement to benefits on June 7, 1999 (DX 25). The Employer contested the award on June 24, 1999, and thereafter submitted additional medical evidence (DX 26). The District Director again granted benefits on October 5, 1999 (DX 34). The Employer appealed on October 27, 1999, and requested a hearing (DX 36). This matter was referred to the Office

Hearing is indicated as "TR", Claimant's Exhibits are indicated as "CX", and Employer's Exhibits are indicated as "EX."

² Various physicians use the terms "stroke," "cerebrovascular accident," and "CVA" interchangeably.

of Administrative Law Judges on December 13, 1999 (DX 45).

A hearing was held in Beckley, West Virginia on September 19, 2000, at which all parties were afforded a full opportunity to present evidence and argument. Director's Exhibits one (1) through forty-six (46), Claimant's Exhibits one (1) through four (4), and Employer's Exhibits one (1) through eighteen (18) were received into evidence (TR 7, 34, 48). Employer was given thirty days from the date of the hearing in which to respond to the report of Dr. Steven M. Koenig (TR 47). Claimant was given thirty days in which to respond to the reports of Drs. Spagnolo and Stewart³ (TR 48). Claimant submitted Dr. Koenig's supplemental report dated October 18, 2000, on October 19, 2000, and the same is hereby admitted into evidence. The record is now closed

ISSUES

- (1) Whether Claimant has two dependents for purposes of augmentation of benefits;
- (2) Whether Claimant has proved that he has complicated pneumoconiosis;
- (3) Whether Claimant has proved that he is totally disabled due to pneumoconiosis; and
- (4) Whether Claimant has proved a material change in conditions since his last claim was denied.

FINDINGS OF FACT, DISCUSSION, AND CONCLUSIONS OF LAW

Length of Coal Mine Employment and Responsible Operator

The Claimant alleged thirty-one years of coal mine employment, and the Employer stipulated to fifteen years, as found by Administrative Law Judge Martin J. Dolan, Jr. in his October 6, 1992 Decision and Order, and as affirmed by the Benefits Review Board as unchallenged (TR 7). After Claimant testified that Westmoreland Coal Company was his last coal mine employer, for whom he worked full-time for thirteen years, Employer conceded that it is the responsible operator (TR 8, 12, 26-27). Once Claimant suffered his stroke in 1981, he ceased all work (TR 13). Claimant testified that all of his coal mine employment was underground and dusty (TR 13). He worked primarily as a supplier which required heavy manual labor (TR 13).

Claimant testified that his first coal mine employer was Wally [sic] Wyoming, for whom he worked thirteen or fourteen years (TR 12). At hearing, he did not recall any other coal mine employment. However, on his Employment History form, Claimant indicated that he worked for Raleigh-Wyoming Coal Company from 1947 to 1962, Mary Frances Coal Company from 1962 to 1965, and Westmoreland from

³ Although the cover letter to EX 18 states that it includes a September 11, 2000 supplemental report from Dr. Thomas M. Jarboe, the report is in fact from Dr. Bruce N. Stewart.

1965 to 1981, for a total of thirty-four years (DX 2). The Social Security Itemized Statement of Earnings confirms a total of thirty-one years of coal mine employment: thirteen and three-quarters years of coal mine employment from 1946 through 1960 with Raleigh Wyoming Mining Company; one additional quarter in 1960 with Polly Collins Coal Company; one and one-quarter years of coal mine employment with Mary Frances Coal Company from 1961 through 1963; five and three-quarters years of coal mine employment with Winding Gulf Coals Inc. from 1965 through 1970; and ten years of coal mine employment with Westmoreland from 1971 through 1980 (DX 4). Accordingly, based on the testimony of the Claimant, the Employer stipulations, and the Social Security records, Claimant is credited thirty-one years of coal mine employment, and Westmoreland Coal Company is the properly named responsible operator.

Background

Claimant was born on June 6, 1927 (DX 1). He claims three dependents: his wife, Frances Oliver Carson, whom he married on June 7, 1947; his adopted grandson, Derek Ramon Carson, born August 22, 1985; and his eleven year old grandson Darelle, born February 2, 1989 (DX 1; TR 19-20, 30). Claimant testified that both Derek and Darelle live with him and his wife and depend on him for their food, clothing, and shelter, as no one else pays for any of these essentials (TR 20, 23). The Claimant stated, and the record confirms, that he and his wife officially adopted Derek, while Darelle has lived with them since he was a baby (DX 8; TR 22). Mrs. Carson also testified that the boys' biological mother is Stephanie Carson, who moved to Florida (TR 31). She stated that Darelle visits his mother in the summer, but she does not support him financially (TR 31). Based on the testimony of the Claimant and his wife and the record, this tribunal finds that Claimant has established two dependents for purposes of augmentation of benefits: his wife and son, Derek. Darelle does not meet the definition of a child under §725.208, and, therefore, cannot be deemed the claimant's dependent child under §725.209.

Claimant testified that he began smoking in 1951 at a rate of one-third pack of cigarettes a day at first, and that he currently smokes two to three cigarettes a day (TR 23, 27). He further said that when he went to see Dr. Zaldivar in 1999, his son, who drove him to the appointment, was smoking. Claimant uses a cane to walk short distances but a wheelchair for long distances (TR 25). He has to stop often to catch his breath. Mrs. Carson testified that she noticed her husband was short of breath before he ever suffered his stroke (TR 29). He becomes dyspneic when he is agitated, nervous, or moves too fast (TR 30). He sleeps on two or three pillows so he can breathe at night (TR 30).

In November 1981, Claimant had a stroke. Pulmonary function studies performed prior to the stroke indicated a mild restrictive impairment (DX 43-49; CX 2). No etiology was attributed to the relatively minimal impairment (DX 43-49). The impairment progressed to a moderate restrictive impairment in the late 1980s (CX 2). By 1990, Claimant's impairment was primarily obstructive (CX 2). When they affect lung function, strokes immediately cause a restrictive effect that improves or dissipates over time (CX 3). This progression may account for the observed change in Claimant's pulmonary impairment from primarily restrictive to primarily obstructive.

Medical Evidence

The medical evidence relevant to the issues of complicated pneumoconiosis and total disability due to pneumoconiosis, submitted since the last denial of October 6, 1992, is listed below. Because this is a duplicate claim, this tribunal considers evidence in existence at the time the prior claims only to the extent necessary to determine whether there has been a material change in conditions since those denials. *Cline v. Westmoreland Coal Co.*, 21 B.L.R. 1-69 (1997).

Chest X-ray Evidence⁴

<u>Exh. No.</u>	<u>Date of X-ray</u>	<u>Date of Report</u>	<u>Physician/ Qualifications</u>	<u>Diagnosis</u>
DX 15	1/20/99	2/1/99	Patel, B/R	1/2; q/r; 4 zones; size A large opacity in left upper lung zone; coalescence; emphysema
DX 14	1/20/99	2/26/99	Navani, B/R	1/1; r/q; coalescence; tuberculosis
DX 13	1/20/99	3/10/99	Ranavaya, B	1/2; q/r; 6 zones; coalescence
CX 2	1/20/99	5/9/00	Cohen, B	2/2; r/q; 6 zones; coalescence
CX 4	1/20/99	8/14/00	Alexander, B/R	2/1; r/q; 4 zones; areas of coalescence in both upper zones; emphysema
DX 33	1/20/99	8/26/99	Wiot, B/R	1/2; q/t; 4 zones; coalescence of pneumoconiotic nodules on the left
DX 33	1/20/99	9/09/99	Meyer, B /R	1/1; r/q; 3 zones; focal areas of coalescence in a perihilar distribution in both upper lung zones
DX 35	9/08/99	9/28/99	Zaldivar, B	1/1; q/r; 4 zones; emphysema
DX 38	9/08/99	10/21/99	Wiot, B/R	1/2; q/t; 2 zones; stranding extending from the left upper lung field toward the left hilum;

⁴ The following abbreviations are used in describing the qualifications of the physicians: B = B-reader and R = Board-certified radiologist. Although the credentials of these physicians may not all be in the record, judicial notice of their qualifications is taken in accordance with www.ABMS.org and the 2000 NIOSH B-reader list. See *Maddaleni v. Pittsburgh & Midway Coal Mining Co.*, 14 B.L.R. 1-135 (1990).

<u>Exh. No.</u>	<u>Date of X-ray</u>	<u>Date of Report</u>	<u>Physician/Qualifications</u>	<u>Diagnosis</u>
				coalescence of pneumoconiotic nodules on the left; emphysema
DX 42	9/08/99	11/07/99	Spitz, B/R	1/2; q/q; 2 zones; coalescence of nodules on the left; emphysema; coalescence of nodules on the left; emphysema
EX 4	9/08/99	11/30/99	Meyer, B /R	1/2; r/q; 3 zones; nodular opacities in the apices bilaterally demonstrate coalescence in the left apex; emphysema
EX 1	9/08/99	1/28/00	Castle, B	1/1; q/r; 2 zones;
EX 5	9/08/99	4/17/00	Binns, B/R	1/1; q/s; 4 zones; some pneumoconiosis and t opacities are seen; emphysema
EX 5	9/08/99	4/19/00	Abramowitz, B/R	2/1; q/r; 4 zones; hyperinflated lungs
EX 5	9/08/99	4/20/00	Gogineni, B/R	1/1; q/r; 5 zones; coalescence; emphysema
EX 5	9/08/99	4/25/00	Baek, B/R	2/1; q/t; 4 zones; hyperinflation
CX 2	9/08/99	5/9/00	Cohen, B	1/2; r/q; 6 zones; coalescence
CX 4	9/08/99	8/14/00	Alexander, B/R	2/1; q/r; 4 zones; areas of coalescence in both upper zones; emphysema

Pulmonary Function Studies

<u>Exh. No.</u>	<u>Test Date</u>	<u>Doctor</u>	<u>Co-op/Undstd/TR⁵</u>	<u>FEV1</u>	<u>FVC</u>	<u>MVV</u>	<u>Qual.⁶</u>	<u>Hgt</u>
DX 10	1/20/99	Rasmussen	Good/Good/Yes	1.24	2.18	45	Yes	67"
				1.43	2.06	53	Yes	

Validation: Dr. M. I. Ranavaya found the pre-bronchodilator study valid but did not review the post-

⁵ Conforming reports of pulmonary function studies must record the miner's level of cooperation and understanding of the procedures, and include three tracings of the maneuvers performed.

⁶ Values listed are those values obtained pre-bronchodilator. The second line of values shown for the January 20, 1999 study indicates a post-bronchodilator study.

bronchodilator study (DX 23). Dr. Ranavaya is board-certified in occupational medicine.

DX 35	9/08/99	Zaldivar	-----/-----/Yes	1.22	2.03	38	Yes	67"
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Validation: Dr. Zaldivar found the study valid. Dr. Stewart found that the validity of this study is questionable (EX 4).

Dr. Spagnolo found both studies to be unreliable based on Claimant's poor muscle strength secondary to his stroke (EX 3). Dr. Koenig found both studies to be valid and reliable based on their acceptability, reproducibility and selection, and found them technically valid based on American Thoracic Society guidelines (CX 3, 5).

Arterial Blood Gas Studies

<u>Exh. No.</u>	<u>Test Date</u>	<u>Doctor</u>	<u>Condition</u>	<u>pCO2</u>	<u>pO2</u>	<u>Alt.</u>	<u>Qualify</u>
DX 12	1/20/99	Rasmussen	resting	39.0	65.0	0-2999	No
DX 35	9/08/99	Zaldivar	resting	39.0	75.0	0-2999	No

Medical Reports/Opinions

The record contains three pages of miscellaneous medical records from Raleigh Regional Cancer Center (EX 2). The first page, dated January 25, 1995, contains progress notes from Dr. Carl S. Larson. Dr. Larson stated that he had not seen the Claimant for more than a year and that he had treated the Claimant for hypertension and complications related to a stroke, with left hemiparesis. Dr. Larson saw the Claimant on this date for a fall which may have caused a fracture of the left hip. The second page, dated October 12, 1995, appears to be a follow-up by Dr. Rajiv Khanna for assessment of the left hip fracture, hypertension, left hemiparesis secondary to old stroke and history of seizure disorder. The Claimant underwent left hip pinning in January 1995 and remained wheelchair bound secondary to the hemiparesis, although he is able to walk with the help of a walker. The third page, dated November 24, 1998, is a progress report from Dr. Larson. Dr. Larson stated that he had not seen the Claimant for more than three years. He noted a history of hypertension and massive CVA which had left the Claimant quadriplegic.

Dr. D.L. Rasmussen, who is board-certified in internal medicine, examined the Claimant on January 20, 1999 (DX 11). He considered thirty-six years of coal mine employment, lastly as a main line motor and beltman requiring heavy lifting and shoveling; a medical history most significant for a CVA in 1981; a history of smoking one-half pack of cigarettes a day for forty-seven years before quitting in 1997; and presenting symptoms of wheezing, a productive cough, shortness of breath, orthopnea, and ankle edema. Dr. Rasmussen also reviewed the results of an x-ray, pulmonary function study, blood gas study, and

physical examination which showed reduced lung expansion, minimally reduced breath sounds, and transient rhonchi on the right. Dr. Rasmussen diagnosed complicated coal workers' pneumoconiosis based on the x-ray and over thirty-one years of coal mine employment, and chronic obstructive pulmonary disease based on airflow obstruction and a productive cough. Accounting for both Claimant's clinical and legal pneumoconiosis, Dr. Rasmussen determined that the pneumoconiosis was caused by coal mine dust exposure, while the chronic obstructive pulmonary disease was due to both coal mine dust exposure and cigarette smoking. He found that overall, the Claimant had minimal lung function loss but would be unable to perform very heavy manual labor.

In a letter dated May 21, 1999, Dr. Rasmussen explained that he re-measured the Claimant and found him to be 66 ½ inches tall in a stooped position (DX 21). After recalculating the pulmonary function studies given the new height, he found that Claimant suffers from severe loss of pulmonary function and is incapable of performing his previous coal mine employment. He noted three risk factors for the impairment: cigarette smoking; coal mine dust exposure; and his left hemiparesis. Dr. Rasmussen opined that the Claimant's coal mine dust exposure played a major role in his disabling respiratory impairment.

Dr. Rasmussen reviewed the reports of Drs. Spagnolo and Morgan on May 12, 2000 (CX 1). Dr. Rasmussen pointed out that the Claimant's FEV1 deteriorated at a rate twice what would be expected between 1989 and 1999, and that neuromuscular weakness generally produces a restrictive defect with a normal FEV1/FVC ratio, which is not present in Claimant's case. For these two reasons, he concluded that Dr. Spagnolo's report is deficient. Regarding Dr. Morgan's report, Dr. Rasmussen stated that coal mine dust exposure is a known cause of chronic obstructive pulmonary disease, citing two medical journal articles in support of this proposition. Dr. Rasmussen further explained that Dr. Morgan's belief that there can be no further progression of pulmonary impairment due to coal mine dust once coal mining ceases is not supported by the literature. He cited several articles in support of his position. Therefore, based on the Claimant's progression of pulmonary impairment which he opined is not solely attributable to Claimant's CVA, and in consideration of Claimant's coal mine employment history, Dr. Rasmussen concluded that both his coal mine dust exposure and cigarette smoke exposure have contributed to his impaired respiratory function.

Dr. George L. Zaldivar examined the Claimant on September 8, 1999 and reviewed additional medical records for a report dated September 28, 1999 (DX 35). He considered a history of working thirty-three years in the coal mines, lastly as a mainline motorman, a medical history, symptoms, including difficulty breathing and muscle weakness, wheezing, difficulty sleeping and some swelling of his feet. He also noted a history of smoking one-half pack of cigarettes every two days for approximately forty years before quitting in 1986 or 1987. Based on a physical examination, pulmonary function study, chest x-ray, and blood gas study, in addition to a review of medical records including the reports of Drs. Chillag, Stewart, Fino, Horsman, Kress, and Rasmussen, Dr. Zaldivar diagnosed simple coal workers'

pneumoconiosis.⁷ He noted that the Claimant has a respiratory impairment, but none of the impairment is due to pneumoconiosis or any chronic dust disease of the lungs caused by his occupation as a coal miner. Dr. Zaldivar concluded that Claimant is disabled due to a stroke and peripheral vascular disease. He also noted that the Claimant continues to smoke which has caused some damage to the lungs. Dr. Zaldivar is board-certified in internal medicine, pulmonary diseases and critical care medicine.

On July 11, 2000, Dr. Zaldivar reviewed medical evidence submitted for the record since his examination of Claimant on August 16, 1989 (EX 11). Based on his review of seven chest x-ray readings, the medical reports of Drs. Chillag, Stewart, Castle, Cohen, Morgan, Spagnolo, and Rasmussen, and a deposition of Dr. Wiot taken on May 31, 2000, Dr. Zaldivar found sufficient evidence to justify a diagnosis of coal workers' pneumoconiosis. He noted that the Claimant has a pulmonary impairment, but none of the impairment is due to pneumoconiosis. Instead, he opined that the impairment is due to the cerebrovascular accident with hemiparesis. From a pulmonary standpoint, Claimant is permanently and totally disabled and unable to perform his usual coal mining job. However, Dr. Zaldivar concluded that none of the disability is due to coal workers' pneumoconiosis. He also noted that there has been some change in condition since 1992 in that the Claimant has developed more airway obstruction due solely to an ongoing smoking habit. Dr. Zaldivar contradicted Dr. Cohen's determination that Claimant's stroke did not cause his obstructive impairment by citing a medical study of eight patients with hemiparesis and comparing that study's findings pursuant to observed vital capacity, residual volume and total lung capacity to Claimant's observed pulmonary function testing. Based on his comparison, Dr. Zaldivar determined that all of Claimant's impairment is due to the stroke.

On September 12, 2000, Dr. Zaldivar was deposed (EX 17). After providing his credentials, Dr. Zaldivar summarized the findings of his prior medical examination of the Claimant in September of 1999. Based on his interpretation of films, Dr. Zaldivar found no progression of the x-ray abnormalities between 1989 and 1999. He did find evidence of simple coal workers' pneumoconiosis and that the Claimant suffers pulmonary impairment. Dr. Zaldivar related the impairment not to the lungs themselves, but to weakness caused by a stroke and cigarette smoking. He found no disability resulting from coal dust exposure. Dr. Zaldivar stated that he reviewed additional medical opinions and specifically disagreed with Dr. Cohen's assessment of severe obstructive lung disease when the Claimant left the mines due to the invalidity of the ventilatory test results upon which he based his opinion. Dr. Zaldivar also disagreed with Dr. Koenig's assessment that coal mine dust induced lung disease could account for the Claimant's obstructive impairment after he left the mines. Dr. Zaldivar found no obstruction when the Claimant quit work in the mines or when he examined the Claimant in 1989. Instead, Dr. Zaldivar concluded that any obstruction was caused by the stroke and the Claimant's continued smoking. Dr. Zaldivar also concluded that Claimant does not have a restrictive impairment because his total lung capacity is normal, and attributed Claimant's reduced FVC to weakness in the chest muscles due to the stroke. (EX 17, p. 37).

⁷ Although "coal workers' pneumoconiosis" may be used synonymously with pneumoconiosis in medical circles, the two terms are distinct legally." *Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819, 821 (4th Cir. 1995).

On November 10, 1999, Dr. Shawn Chillag, who is board-certified in internal medicine and geriatrics, reviewed the medical reports of Drs. Stewart, Fino, Rasmussen, and Zaldivar, in addition to a deposition of Dr. Fino and three chest x-rays (DX 39). Based on his review of this evidence, he concluded that there is sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis, and that the Claimant has pulmonary impairment related to a stroke and possibly cigarette smoking. In his opinion, the Claimant is totally and permanently disabled due to his stroke and vascular disease which are unrelated to his simple coal workers' pneumoconiosis. Dr. Chillag does not believe there has been any material change in the Claimant's pulmonary function since 1992.

Dr. Chillag reviewed additional medical evidence on June 29, 2000 (EX 7). Dr. Chillag reviewed the medical reports of Drs. Fino, Cohen, Stewart, Castle, Morgan, Spagnolo, and Rasmussen, in addition to the deposition of Dr. Wiot. He also reviewed the results of two chest x-rays from September 8, 1999 and September 20, 1999. Dr. Chillag pointed out that because of the Claimant's stroke and left sided paralysis, pulmonary testing has become difficult and problematic to quantify. He believes that Claimant does have some pulmonary impairment related to muscular weakness from his stroke and cigarette smoking. He found the Claimant is unable to do his regular coal mining job. In his opinion, the Claimant's total disability is unrelated to his simple coal workers' pneumoconiosis. On August 28, 2000, Dr. Chillag affirmed his June 29, 2000 medical opinions after reviewing Dr. Koenig's August 21, 2000 medical report and two chest x-rays (EX 15).

On November 23, 1999, Dr. Bruce N. Stewart reviewed specified medical evidence (EX 4). He reviewed a history of thirty-one years of coal mine employment, lastly as a track man and motorman, a history of smoking approximately one-half pack of cigarettes beginning at age twenty-two and quitting in 1990, a medical history, ten x-ray readings of three chest x-rays, two pulmonary function studies dated January 20, 1999 and September 8, 1999, and two blood gas studies of the same dates. Dr. Stewart also reviewed the medical reports and depositions of Drs. Rasmussen, Zaldivar, Chillag, and Fino. Based upon his review of the medical evidence, Dr. Stewart found sufficient evidence to justify a diagnosis of coal workers' pneumoconiosis. He also found that Claimant does have respiratory impairment, and agreed with Drs. Fino and Zaldivar that the most likely cause of this impairment was the Claimant's stroke in 1981. Dr. Stewart concluded that the Claimant is totally and permanently disabled secondary to the crippling stroke that occurred in 1981 leaving him with paralysis of the left arm and leg. He found no evidence of a disabling lung disease and further opined that if the Claimant had not had the stroke, he would have retained sufficient lung capacity to continue his prior mining duties. Dr. Stewart is board-certified in internal medicine and pulmonary disease.

Dr. Stewart reviewed additional medical evidence on July 6, 2000 (EX 9). The new evidence included nine readings of two chest x-rays, the deposition of Dr. Wiot, and the medical reports of Drs. Chillag, Fino, Castle, Cohen, Morgan, Spagnolo and Rasmussen. After reviewing the above medical evidence, Dr. Stewart affirmed his diagnosis of simple coal workers' pneumoconiosis based on a history of working in the coal mines and chest x-ray reports. His opinion that Claimant did not have any significant respiratory impairment prior to his stroke remained unchanged. He concluded that there is no objective

evidence to suggest that there is any obstructive lung disease caused by coal workers' pneumoconiosis. Dr. Stewart concluded that there has been no significant material change in Claimant's condition since 1992 and that Claimant is totally and permanently disabled due to his stroke, not due to coal workers' pneumoconiosis or smoking.

Dr. Stewart reviewed Dr. Koenig's August 21, 2000 medical report on September 11, 2000, and stated that his prior medical opinions remain unchanged (EX 18). He did add, however, that Claimant has experienced a decrease in lung function since 1992. He attributed this change to a continued smoking habit, which has led to the progression of chronic obstructive pulmonary disease. He reiterated that the change was not due to coal workers' pneumoconiosis or coal dust inhalation. Dr. Stewart agrees that pneumoconiosis can cause obstructive lung disease, but does not feel it is the case here. Although he believes that pneumoconiosis can progress after leaving the coal mines, he does not believe it would cause the loss of lung function demonstrated in Claimant's case.

Dr. Gregory J. Fino reviewed specified medical evidence on November 23, 1999 (DX 40). He considered a medical history; a history of thirty-one years of coal mine employment, lastly as a track man, motorman and mainline motorman, and a smoking history of one package of cigarettes per day for many years before quitting in 1981. He also reviewed the medical reports of Drs. Chillag, Zaldivar, Fino, and Rasmussen, and the results of eleven readings of three chest x-rays, six pulmonary function studies and four blood gas studies. Dr. Fino diagnosed simple coal workers' pneumoconiosis. He noted that there is respiratory impairment present due to weakness caused by a stroke and suspected smoking. From a respiratory standpoint, Dr. Fino stated that the Claimant is disabled from returning to his last mining job or a job requiring similar effort. But, Dr. Fino concluded that coal mine dust inhalation neither caused nor contributed to this disability. The Claimant, he opined, would be as disabled had he never stepped foot in the mines. Dr. Fino is board-certified in internal medicine and pulmonary disease.

On July 20, 2000, Dr. Fino reviewed additional medical records (EX 12). Dr. Fino considered eleven readings of two chest x-rays, the medical reports of Drs. Chillag, Stewart, Castle, Cohen, Morgan, Spagnolo, Rasmussen and Wiot, two pulmonary function studies and two blood gas studies. Based on his review of the additional medical evidence, Dr. Fino stated his prior medical opinions remain unchanged. He also reviewed a number of articles regarding coal mine dust inhalation and obstructive lung disease as cited by Drs. Cohen and Rasmussen. He noted that there is a difference between clinical significance and statistical significance. In his opinion, the articles show a statistically significant obstructive abnormality in some miners, but no clinical significance. Moreover, according to his interpretation, none of the articles show that the obstruction causes a clinically significant impairment or disability.

Dr. Fino prepared a supplemental report on August 30, 2000, based on his review of Dr. Koenig's August 21, 2000 report (EX 13). The medical report did not change his opinion that Claimant has simple coal workers' pneumoconiosis, and that the Claimant's disabling respiratory impairment developed due to weakness from a stroke and suspected smoking.

Dr. James R. Castle reviewed medical evidence on December 6, 1999 (EX 4). He considered thirty-three years of coal mine employment in the underground mines as a track man and motorman, a history of smoking one-half to one pack of cigarettes per day for twenty years, a medical history, nineteen reports of eight separate x-rays, ten pulmonary function studies, and eight blood gas studies. Dr. Castle also considered the medical reports of Drs. Larson, Horsman, Kress, Daniel, Zaldivar, Chillag, Stewart, Fino, and Rasmussen. He found radiographic evidence of simple coal workers' pneumoconiosis, but no evidence of complicated pneumoconiosis. He diagnosed mild obstructive airway disease as a result of a long and extensive tobacco smoking habit. He found Claimant to have mild respiratory impairment based on the obstructive airways disease and muscular weakness due to previous cerebrovascular accident. In his opinion, the Claimant retains the respiratory capacity to perform his usual coal mining employment duties. Dr. Castle further found that the Claimant is not permanently and totally disabled as a result of coal workers' pneumoconiosis or any other process arising from his coal mining employment. He did find that Claimant is permanently and totally disabled as a result of his previous cerebrovascular accident and severe peripheral vascular disease, both diseases of the general public and unrelated to coal mining employment or coal dust exposure. Dr. Castle is board-certified in internal medicine and pulmonary disease.

Dr. Castle also reviewed additional medical evidence on July 14, 2000 (EX 10). His review included medical reviews by Drs. Chillag, Fino, Stewart, Cohen, Morgan, Spagnolo, and Rasmussen, a deposition of Dr. Wiot on May 31, 2000, and six readings of a September 8, 1999 chest x-ray. From his review of this additional medical evidence, Dr. Castle stated that nothing altered any of his opinions previously stated in his December 6, 1999 report.

In a supplemental report dated August 30, 2000, Dr. Castle stated, based upon his review of Dr. Koenig's report dated August 21, 2000, that his original opinions remain entirely unchanged (EX 14). Dr. Castle opined that the miner's elevated carboxyhemoglobin level showed that he was still smoking, and stated that even if he were smoking one-half pack a day, giving him at least a twenty-five pack-year history, it would be significant enough to develop chronic obstructive pulmonary disease. He noted a normal diffusing capacity after correction for alveolar volume. In his opinion, Dr. Zaldivar's pulmonary function study showed a normal total lung capacity and no evidence of a restrictive lung disease, clearly indicating that the reduction in FVC and FEV1 were primarily related to muscle weakness rather than intrinsic lung disease. In his opinion, the Claimant would not have normal total lung capacity if he had restrictive lung disease due to pneumoconiosis. He found a mild obstructive lung disease which manifested eighteen years after he ceased mining, and because there was no corresponding progression of the pneumoconiosis by x-ray, Dr. Castle felt that ongoing cigarette smoking is the cause of the obstructive defect. Dr. Castle agrees with Drs. Morgan, Fino, and Zaldivar that the airway obstruction is related to smoking. Dr. Castle disagrees with Dr. Koenig's reasoning. He reaffirmed that the Claimant's mild emphysema is due to his ongoing and extensive smoking history.

Dr. Castle was deposed on August 30, 2000 (EX 16). Dr. Castle summarized his medical credentials and affirmed his findings as provided in his medical reports dated December 6, 1999, July 14, 2000 and August 30, 2000. Dr. Castle stated that he also had an opportunity to review additional medical

reports by Drs. Chillag, Morgan, Stewart, Spagnolo, Zaldivar, Fino and Koenig. After reviewing the additional reports, Dr. Castle disagreed with Dr. Koenig's finding that the Claimant's impairment was due to both coal dust exposure and cigarette smoking. Dr. Castle opined that the medical evidence clearly shows that Claimant did not have a disabling respiratory impairment when he left the mining industry and had a cerebrovascular accident. He pointed out that the Claimant's coal workers' pneumoconiosis has not at all progressed radiographically since he left the coal mines, but the development of a mild to moderate degree of airway obstruction and pulmonary emphysema have developed.⁸ Dr. Castle concludes that these conditions are not related to anything but an ongoing smoking habit.

Dr. Wiot, who is a board-certified radiologist, was deposed on May 31, 2000, at which time he provided his credentials, reiterated the results of his medical review, and discussed other evidence (EX 6). Based on his review of x-rays dated August 16, 1989, September 8, 1999 and January 20, 1999, Dr. Wiot found evidence that Claimant has simple coal workers' pneumoconiosis. He did not find evidence of complicated coal workers' pneumoconiosis. Dr. Wiot explained the difference between coalescence, which he observed in Claimant's x-rays, and lesions of complicated pneumoconiosis (EX 6, p. 29-30).

On March 19, 2000, Dr. W.K.C. Morgan reviewed medical evidence (EX 3). He considered the medical reports of Drs. Rasmussen, Bassali, Horsman, Larson, Previll, Hayes, Daniel, Kress, Zaldivar, Chillag, Stewart, Fino, Patel, Meyer, Wiot, and Castle, which included the results of chest x-rays, pulmonary function studies and blood gas studies; a history of smoking one-half pack of cigarettes a day for twenty years; and thirty-four to thirty-five years of coal mine employment as a motorman. Dr. Morgan found sufficient evidence to justify a diagnosis of some form of pneumoconiosis induced by his work in the coal mines. However, he opined that the pneumoconiosis has not produced any pulmonary or respiratory impairment. In his opinion, Claimant is totally disabled on account of a stroke he suffered in 1981 after leaving his work in the coal mines, which prevents him from working. Dr. Morgan found mild to moderate airways obstruction as a result of the Claimant's smoking habit.

Dr. Morgan reviewed additional evidence on July 2, 2000, including the reports of Drs. Cohen, Spagnolo, and Rasmussen, a chest x-ray reading of September 8, 1999, and a deposition taken of Dr. Wiot on May 31, 2000 (EX 8). He felt the additional data confirmed his opinion that although Claimant has simple coal workers' pneumoconiosis, his respiratory impairment is related to his stroke and cigarette smoking. He stated that the Claimant's condition has materially changed since 1992, as his lung function has declined. However, he reaffirmed his earlier medical opinions in stating that the decline in lung function is a direct consequence of the Claimant's stroke and cigarette smoking, not exposure to coal dust.

Dr. Samuel V. Spagnolo reviewed specified medical evidence on March 19, 2000 (EX 3). He considered a medical history, symptoms, including shortness of breath, coughing and some dizziness, a

⁸ Dr. Castle acknowledged that while several other interpreting radiologists and B-readers found coalescence, he simply does not consider coalescence a progression (Ex 16, p. 37).

history of thirty-one years of coal mine employment, lastly as a mainline motorman, and a smoking history of one-half package of cigarettes per day for approximately twenty years. He also reviewed the medical reports of Drs. Rasmussen, Horsman, Hayes, Zaldivar, Daniel, Kress, Chillag, Fino, Stewart, and Castle, which included the results of chest x-rays, blood gas studies and pulmonary function studies. Dr. Spagnolo found evidence of simple coal workers' pneumoconiosis, but opined that the Claimant does not have any pulmonary or respiratory impairment attributable to pneumoconiosis or coal dust exposure related to his prior coal mining job. He opined that Claimant could have performed his prior coal mine job before his stroke. Dr. Spagnolo noted that the Claimant's 1981 stroke resulted in severe weakness of the entire left side of the Claimant's body. He opined that Claimant's stroke, not his lung function, is the cause of the Claimant's inability to perform heavy labor or perform his prior coal mining job. Dr. Spagnolo is board-certified in internal medicine and pulmonary diseases.

Dr. Spagnolo reviewed additional evidence on July 9, 2000 consisting of medical reports from Drs. Morgan, Cohen and Rasmussen, a May 31, 2000 deposition of Dr. Wiot and six additional chest x-rays (EX 10). Dr. Spagnolo stated that his prior opinion concerning Claimant remained unchanged. Although the Claimant has simple coal workers' pneumoconiosis, Dr. Spagnolo affirmed his earlier medical opinion that any impairment would be the result of the 1981 stroke.

On September 9, 2000, Dr. Spagnolo provided a supplemental report based on a review of a medical report of Dr. Koenig and two additional chest x-rays (EX 18). He concluded that Claimant has simple coal workers' pneumoconiosis, but does not have a pulmonary or respiratory impairment attributable to a pneumoconiosis. He further found that neither the Claimant's coal dust exposure nor his coal workers' pneumoconiosis have contributed to his current medical conditions. Dr. Spagnolo disputed the validity of the pulmonary function test results relied upon by Dr. Koenig, and therefore he would not agree with Dr. Koenig's assessment of the Claimant. Based on the lack of clinical lung disease by physical examination and normal lung function and blood gas values obtained in 1980, Dr. Spagnolo stated that Claimant did not have a respiratory impairment prior to his stroke in November of 1981.

On January 12, 2000, Dr. Robert Cohen reviewed medical records, including numerous x-ray interpretations from 1979 through 1999, the January 20, 1999 and September 8, 1999 x-rays, and the reports of Drs. Stewart, Fino, Chillag, Kayi, Zaldivar, Rasmussen, Meyer, Castle, Morgan, and Spagnolo (CX 2). After considering the Claimant's symptoms, employment history, medical history, and smoking history of one-half to one pack of cigarettes a day for forty-seven years, ending in 1987 but continuing to smoke at the rate of one to two cigarettes per week, Dr. Cohen opined that the Claimant suffers from simple pneumoconiosis. He further opined that the Claimant's thirty-three years of coal mine employment, along with his twenty-four to forty-eight pack-years of smoking, significantly contributed to the development of his severe obstructive lung disease and severe diffusion impairment. He explained that the Claimant's pulmonary impairment clearly progressed from mild to severe, as did his diffusion impairment, which progressed from 74% to 33% of predicted values on his most recent pulmonary function test, thus disabling him from his last coal mine job, which required extensive heavy lifting.

Dr. Cohen alluded to medical literature in support of the proposition that coal dust exposure can cause obstructive pulmonary disease and lead to significant impairment. He specifically cited a study that showed one pack year of smoking causes a degree of impairment similar to one year of underground coal dust exposure. Dr. Cohen declared that Dr. Morgan's opinion that Claimant's pulmonary impairment cannot be due to coal dust exposure because it progressed after mining stopped is contrary to the medical literature. Therefore, he opined that progression of disease after exposure ceases does not rule out coal dust as a cause of impairment. Dr. Cohen ruled out Claimant's stroke as the cause of Claimant's disabling and primarily obstructive impairment. He noted that Claimant's impairment is mainly obstructive in nature and that he had significant diffusion impairment. Dr. Cohen then explained that the pattern of diffusion impairment was consistent with an altered gas exchanging surface, which is a pattern that can be seen in interstitial lung disease and emphysema, but is not one that is caused by neuromuscular disease such as a stroke. He also contradicted the contrary finding of Drs. Spagnolo, Fino, Zaldivar, Kress, and Chillag, who all concluded that Claimant's stroke was the cause of his impairment, by explaining that "even though stroke can cause a restrictive impairment with low diffusion, the associated diffusion impairment with stroke is due to loss of volume, and has a normal D1/Va. Mr. Carson had a low D1/Va measurement on diffusion capacity." Dr. Cohen is board-certified in internal medicine and pulmonary disease.

On August 21, 2000, Dr. Steven M. Koenig, who is board-certified in internal medicine and pulmonary disease, reviewed medical evidence (CX 3). He considered a smoking history spanning thirty-seven years at a rate of one-half to one pack of cigarettes per day, and a continued habit of smoking one or two cigarettes per week. Dr. Koenig considered an employment history of approximately thirty-five years, a medical history, x-ray reports, pulmonary function and blood gas studies, and the medical reports. Dr. Koenig determined that all of the pulmonary function studies, focusing his exposition on the tests performed on August 16, 1989, January 20, 1999, and September 8, 1999, are valid based on their acceptability, reproducibility, and selection. He noted that the January 1 and September 8, 1999 studies are representative of Claimant's lung function because they are almost identical. Dr. Koenig concluded that the Claimant's coal dust exposure was sufficient to cause respiratory impairment in a susceptible host. He found severe respiratory impairment due to obstructive lung disease and respiratory muscle loss. He opined that the former is due to chronic obstructive pulmonary disease, including chronic bronchitis and emphysema, and the latter is due to a cerebrovascular accident.

Dr. Koenig pointed out that pulmonary impairment secondary to a stroke results in a restrictive impairment, while COPD leads to an obstructive impairment. He referred to a significant decrease in the Claimant's lung function between eight and eighteen years after his stroke and explained that because a stroke exerts its maximum effect on lung function shortly thereafter, and usually improves with time, this later decline in pulmonary function cannot have been caused by the CVA. Therefore, he disagreed with the opinions of Drs. Stewart and Spagnolo who determined that all of Claimant's impairment was due to his stroke. Based on a valid 1999 pulmonary function study, he also disagreed with Dr. Stewart's statement that there has been no appreciable change in the miner's condition since 1992. Dr. Koenig believed that the COPD would render Claimant totally disabled even if he had never suffered the stroke. While Dr. Koenig admitted that cigarette smoking may have contributed to the COPD and consequent respiratory

impairment, he felt the coal dust exposure alone could also have accounted for, or at least significantly contributed to, Claimant's severe respiratory impairment.

Dr. Koenig stated, "The symptoms, pulmonary function tests and chest x-ray appearance of COPD are identical whether caused by coal dust, by cigarette smoke, or by both. Thus, when a miner who has significant exposure to coal dust and to cigarette smoke develops COPD, it is often impossible to determine whether coal dust or cigarette smoke causes the COPD exclusively. In such circumstances, the only sound medical diagnosis is that neither can be excluded as a cause, so both must be included as a cause." His conclusions are based on numerous studies in medical literature that he, as well as other pulmonary experts and NIOSH, found methodologically valid, and which he believes Drs. Fino and Zaldivar ignored.

Dr. Koenig provided a supplemental opinion on October 18, 2000 (CX 5). He considered the reports of Drs. Chillag, Spagnolo, Fino, and Stewart. He agreed that Claimant has obstructive pulmonary function test results, but disagreed with Dr. Stewart about the validity of the Claimant's pulmonary function studies. Dr. Koenig found the pulmonary function studies valid based on their acceptability, reproducibility, and selection. He noted that Dr. Stewart's only criticism was Dr. Koenig's use of the American Thoracic Society's guidelines, which he, Dr. Koenig, found more scientifically sound than those set by the Code of Federal Regulations. He also asserted that the deterioration of Claimant's post-bronchodilator FVC is not indicative of lack of effort but rather indicative of a patient with asthma and chronic obstructive pulmonary disease.

Dr. Koenig criticized Dr. Spagnolo's critiques of the pulmonary function studies and countered that Dr. Spagnolo's belief that a test can be invalid despite having values within 5% of each other is contrary to medical literature and reasoned medical opinion. He pointed out that this occurred on tests taken eight months apart. Dr. Koenig stated that respiratory muscle weakness results in restrictive, not obstructive, impairment, and since Claimant demonstrated an obstructive impairment, the CVA cannot be the cause thereof.

Dr. Koenig pointed out that a significant decline in lung function occurred many years after the CVA. He opined that the majority of the Claimant's pulmonary impairment and disability is caused by chronic obstructive pulmonary disease, and, even discounting any pulmonary impairment caused by the CVA, Dr. Koenig believes the Claimant would still be totally disabled from a respiratory standpoint. He further opined that, in theory, chronic obstructive pulmonary disease could be due to cigarette smoking, coal dust exposure, or a combination of both. Dr. Koenig referred to "ample evidence in the medical literature that coal-dust induced COPD in non-smokers can be severe and disabling." He stated that in this case it is impossible to determine whether either cause is exclusive, given extensive histories in both smoking and coal mine employment. Therefore, he declared that the sound medical judgment is that neither can be excluded. Dr. Koenig further criticized the reports of Drs. Chillag and Stewart for not citing any medical literature for their position that COPD cannot be secondary to coal dust exposure if it begins years after coal mine employment stops and in the absence of a progression of x-ray abnormalities. Dr. Koenig asserted that the medical literature emphasizes that coal mine dust causes clinically significant obstruction

with or without cigarette smoking.

Duplicate Claim in Fourth Circuit

To assess whether a material change in conditions is established, the Administrative Law Judge must consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proved at least one of the elements of entitlement previously adjudicated against him. *Lisa Lee Mines v. Director, OWCP, (Rutter)*, 86 F.3d 1358, 20 B.L.R. 2-227 (4th Cir. 1996) (*en banc*). Those elements, which Claimant must prove by a preponderance of the evidence in order to establish entitlement to black lung benefits are: (1) the miner has pneumoconiosis; (2) the pneumoconiosis was caused by coal mine employment; (3) the miner is totally disabled; and (4) the miner's disability is caused by pneumoconiosis. *See Trent v. Director, OWCP*, 11 B.L.R. 1-26 (1987); *Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986) (*en banc*). Since the 1988 claim was denied on the basis that Claimant failed to establish that his totally disabling respiratory impairment was due to pneumoconiosis, this tribunal must determine whether the evidence submitted since that denial now establishes that element of entitlement. If it does, Claimant has established a material change in conditions. If it does not, the duplicate claim must be denied.

Complicated Pneumoconiosis and §718.304

Section 718.304 provides an irrebuttable presumption of total disability due to pneumoconiosis if the miner is suffering from a chronic dust disease of the lungs of an advanced degree frequently referred to as complicated pneumoconiosis. *See Usery v. Turner Elkhorn Mining Co.*, 428 U.S. 1,7,11 (1996); *Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250, 255 (4th Cir. 2000). Section 718.304 sets out three manners in which a Claimant may establish the existence of complicated pneumoconiosis: 1) diagnosis by x-ray yielding one or more large opacities classified in Category A, B, or C; 2) diagnosis by biopsy or autopsy yielding massive lesions in the lungs, or 3) when diagnosis by means other than those specified by (a) and (b), would be a condition which could reasonably be expected to yield the results described in paragraph (a) or (b) had diagnosis been made as therein described. Any diagnosis made under paragraph (c) must accord with acceptable medical procedures. §718.304(c). The Board has held that §718.304(a)-(c) does not provide alternative means of establishing the irrebuttable presumption of total disability due to pneumoconiosis, but rather requires the administrative law judge to first evaluate the evidence in each category, and then weigh together the categories at §718.304(a)-(c) prior to invocation. *Melnick v. Consolidation Coal Co.*, 16 B.L.R. 1-31 (1991) (*en banc*); *See also Dennis E. Keene v. G & A Coal Co.*, BRB No. 96-1689 BLA-A (September 27, 1996) (*unpublished*). Since no biopsy or autopsy evidence is of record, this tribunal examined the x-ray evidence for complicated pneumoconiosis under part (a) and equivalent diagnostic evidence under part (c).

Dr. Patel, a B-reader and board-certified radiologist, interpreted the January 20, 1999 x-ray as revealing a size A large opacity classifiable as complicated pneumoconiosis and coalescence of small opacities in the left upper lung zone (DX 15, 16). Drs. Navani, Ranavaya, Cohen, Alexander, Wiot, and

Meyer reread the film, and, although all of them found the existence of either category one or two pneumoconiosis with coalescence, none described the existence of a size A large opacity. Dr. Wiot explained the difference between lesions of complicated pneumoconiosis and coalescence during his May 31, 2000 deposition (EX-6). Dr. Wiot explained that coalescence appears as a “haziness of nodules which have come together,” whereas complicated lesions/opacities are associated with cicatricial emphysema and tend to have an irregular shape in which one cannot see individual nodules. He further explained that the complicated lesion has a different appearance primarily because its pathology consists of “little emphysematous areas” around the opacity which are irregular and have little strands extending out from them. None of the aforementioned physicians commented in regard to whether lesions of complicated pneumoconiosis can form from the coalescence; however, Dr. Wiot distinguished Claimant’s observed coalescence from complicated lesions, explaining that the individual small opacities were visible and there were no visible indications of cicatricial emphysema.⁹

The September 8, 1999 x-ray was interpreted by eleven different physicians, eight of whom are dually qualified board-certified radiologists and B-readers, and none of these physicians found large opacities. Only six physicians observed coalescence. Dr. Castle was not one of those six. In his August 30, 2000 deposition, Dr. Castle explained that lesions of complicated pneumoconiosis can be formed by the coalescence of small opacities (Ex 16). However, he did not opine that this form of progression had occurred in the Claimant’s lungs, nor did any of the other physicians. Even accepting Dr. Castle’s statement that coalescence can result in lesions of complicated pneumoconiosis, because no physician reviewing either the January 20, 1999 or the September 8, 1999 x-rays formed an objective or reasoned opinion that this progression occurred in the Claimant, this tribunal finds that this x-ray evidence, and therefore the x-ray evidence as whole, does not indicate the presence of complicated pneumoconiosis. Since there is no evidence that the coalescence observed is tantamount to a finding of lesions of complicated pneumoconiosis, and several qualified board-certified radiologists and B-readers agree on this point, this tribunal finds that Claimant has not proved the existence of complicated pneumoconiosis under parts (a) or (c) of §718.304. *Cranor v. Peabody Coal Co.*, 22 B.L.R. 1-1 (1999) (*en banc on recon.*); *Sheckler v. Clinchfield Coal Co.*, 7 B.L.R. 1-128 (1984); *Worhach v. Director, OWCP*, 17 B.L.R. 1-105 (1993).

Of the examining physicians, only Dr. Rasmussen diagnosed complicated pneumoconiosis. He based his finding on Dr. Patel’s x-ray interpretation and Claimant’s thirty-one or more years of coal mine employment (DX 11). Because the vast majority of the best qualified physicians did not find complicated pneumoconiosis, because Dr. Rasmussen’s finding is based on the sole x-ray interpretation finding complicated pneumoconiosis, and because none of the other examining or reviewing physicians found complicated pneumoconiosis, this tribunal finds that Claimant has failed to establish the existence of

⁹ Like Dr. Patel, Drs. Navani, Alexander, Wiot, and Meyer are all dually qualified board-certified radiologists and B-readers. Drs. Ranavaya and Cohen are B-readers. Additionally, the record indicates that Drs. Wiot and Meyer are professors of radiology.

complicated pneumoconiosis pursuant to §718.304(c). Accordingly, upon consideration of all of the pertinent evidence regarding Claimant's pneumoconiosis, this tribunal finds that the record does not support a finding that Claimant's pneumoconiosis has advanced to the point where it is classifiable as complicated pneumoconiosis. Therefore, Claimant is not entitled to invoke the irrebuttable presumption of §718.304.

Total Disability and Causation

A claimant must establish, by a preponderance of the evidence, that he is totally disabled due to pneumoconiosis. *See Gee v. W.G. Moore & Sons*, 9 B.L.R. 1-4 (1986). To establish such total disability, a claimant must prove that his pneumoconiosis prevents him from engaging in either his usual coal mine work or comparable and gainful work as defined in Section 718.204. Amended §718.204(c)(1) codifies the relevant case law, and requires the miner to establish that his pneumoconiosis is a substantially contributing cause of his totally disabling respiratory or pulmonary impairment. Pneumoconiosis is a "substantially contributing cause" of the miner's disability if it has a material adverse effect on the miner's respiratory or pulmonary condition, or it materially worsens a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment. §718.204(c)(1)(i) and (ii). A claimant cannot establish eligibility for benefits if he would have been totally disabled "to the same degree [and] by the same time in his life had he never been a miner." *Milburn Colliery Co. v. Hicks*, 138 F.3d 524, 534 (4th Cir. 1998), quoting *Dehue Coal Co. v. Ballard*, 65 F.3d 1189, 1196 (4th Cir. 1995).

Dr. Rasmussen opined that the Claimant's coal mine dust exposure played a major role in his disabling respiratory impairment. Dr. Cohen opined that both the Claimant's coal mine dust exposure and cigarette smoking have contributed to his respiratory impairment. Dr. Koenig asserted that Claimant's coal dust exposure alone could have accounted for, or at least significantly contributed to, his severe respiratory impairment. Dr. Zaldivar opined that none of Claimant's impairment was due to pneumoconiosis or any chronic dust disease of the lungs caused by coal mine employment. He attributed the disability to the Claimant's stroke, peripheral vascular disease, and continued smoking. Dr. Chillag opined that Claimant's pulmonary impairment is related to his stroke and possibly cigarette smoking, but is unrelated to coal workers' pneumoconiosis. Dr. Stewart opined that the Claimant's impairment is most likely due to his stroke and continued smoking. Dr. Fino attributed the Claimant's disability to weakness caused by his stroke and to his suspected smoking. He stated that coal mine dust inhalation neither caused nor contributed to the Claimant's disability. Dr. Castle opined that Claimant is not totally disabled as a result of coal workers' pneumoconiosis. He found only a mild degree of pulmonary emphysema, which he attributed to smoking, and some respiratory impairment due to muscular weakness caused by Claimant's stroke. Dr. Morgan asserted that Claimant is disabled by his stroke, that he has a mild to moderate airways obstruction due to smoking, and that pneumoconiosis has not produced any pulmonary impairment. Dr. Spagnolo opined that the Claimant has no pulmonary or respiratory impairment due to pneumoconiosis; he ascribed the miner's inability to work to his stroke.

Initially, this tribunal recognizes that Drs. Cohen, Koenig, Fino, Stewart, Castle, Spagnolo, and Zaldivar are all board-certified in both internal medicine and pulmonary disease. Dr. Chillag is board-certified in both internal medicine and geriatric medicine, while Dr. Rasmussen is board-certified in internal medicine. Dr. Morgan's certification is from England and Canada, but his curriculum vitae indicates expertise in the field of pulmonary disease. Therefore all of the physicians' opinions merit substantial weight based on their credentials. *Scott v. Mason Coal Co.*, 14 B.L.R. 1-38 (1990); *Wetzel v. Director, OWCP*, 8 B.L.R. 1-139 (1985).

The primary cause for disagreement between Drs. Rasmussen, Cohen, and Koenig, on one side, and Drs. Fino, Stewart, Castle, Spagnolo, Zaldivar, Chillag, and Morgan, on the other side, is that the former physicians have concluded that the Claimant's pneumoconiosis has, in addition to his stroke and smoking history, caused respiratory disability, while the latter doctors reasoned that the stroke, in combination with Claimants smoking habit, is solely responsible for his disability. This disagreement has resulted in an overwhelming number of opinions primarily focused on the body of medical literature related to pneumoconiosis and the etiology of respiratory and pulmonary impairments. While the regulations provide clarification within medical literature and term usage, resolution of the issue of causation depends upon evaluation of the most credible, documented and reasoned medical opinions of record.¹⁰ *Underwood v. Elkay Mining, Inc.*, F.3d 946, 949 (4th Cir. 1997) (The ALJ is entrusted with the authority to "evaluate the evidence, weight it, and draw his own conclusions."); *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 211 (4th Cir. 2000) ("When the ALJ is presented with conflicting medical evidence and conflicting expert opinions, it is the province of the ALJ to evaluate the physicians' opinions.")

The evidence indicates that Claimant suffers from a mild to severe respiratory impairment that is now primarily obstructive in nature and has progressed since Claimant left the mines in 1981, in addition to having category 1 or 2 radiographic evidence of clinical pneumoconiosis. Claimant's impairment has a restrictive element which has largely dissipated since Claimant's stroke and in the presence of the primary obstructive impairment (see CX 2, p. 7-10; EX 17 p. 37-39; DX 43-26). Without discussing etiology, it is important to note that the amended regulations explicitly acknowledge that both forms of impairment are consistent with the conditions classifiable as pneumoconiosis. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment, and includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment. §718.201(a)(2). Furthermore, pneumoconiosis is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure. §718.201(c). By amending the regulations to explicitly include obstructive pulmonary diseases arising out of coal mine employment within the definition of pneumoconiosis, the Department of Labor intended to "eliminate the need for litigation of this issue on a claim-by-claim basis, and render invalid as inconsistent

¹⁰ A "documented" opinion is one that sets forth the clinical findings, observations, facts, and other data upon which the physician based the diagnosis. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation and data adequate to support the physician's conclusions. *Id.*

with the regulations medical opinions which categorically exclude obstructive lung disorders from occupationally-related pathologies” in accordance with well established legal principles. 65 Fed. Reg. 79,938 (December 20, 2000).¹¹

In evaluating the physicians’ opinions, adjudicators must bear in mind that medical professionals generally use medical terms of art, not legal ones. *Compton v. Beth Energy Mines, Inc.*, 1998-B.L.A.-14 (1998) (citing *Roberts v. West Virginia C.W.P. Fund*, 20 B.L.R. 2-69 (4th Cir. 1996). To physicians, “pneumoconiosis” is a single disease, arising in whole from a specific cause (dust exposure), and producing a characteristic form of pulmonary damage. *Id.* To the law, “pneumoconiosis” is an array of diseases or effects, arising in whole or in part from dust exposure, and the form of pulmonary damage is irrelevant, so long as some impairment arises from it. *Id.*

Based on his review of medical records, Dr. Fino determined that Claimant has experienced a decline in lung function since 1981 with a obstructive component that renders him totally disabled. (DX 40). He did not attribute Claimant’s loss in lung function to pneumoconiosis or coal dust inhalation because he maintains that “although coal workers’ pneumoconiosis can be progressive, it is characterized by a worsening chest film.” He therefore concluded that the observed decrease in FEV1 and FVC was attributable to Claimant’s 1981 stroke, which “caused weakened respiratory muscles and is a neuromuscular, not pulmonary, condition.” He later added smoking as an etiologic factor (EX 13). Dr. Fino asserts that it is possible to differentiate the obstruction caused by coal mine dust from the obstruction caused by other factors such as smoking and asthma because the medical literature he credits does not support the conclusion that coal mine dust inhalation causes a clinically significant reduction in FEV1 (EX 12).

In his opinion of July 20, 2000, Dr. Fino reviewed medical literature in support of his opinions. Dr. Fino’s opinions and conclusions drawn from specified medical literature, however, are inconsistent with the findings made by the Department of Labor upon its review of the medical literature and case law in amending the regulations. Moreover, Dr. Fino presented the same opinions to the Department of Labor during the rule making leading up to the amendment of the regulations, and the Department rejected his findings, often stating that his conclusions do not stand up to scrutiny and citing medical literature to the contrary. See 65 Fed. Reg. 79,938 - 79,943 (December 20, 2000). Thus, this tribunal finds that the studies relied upon by Dr. Fino in reaching his conclusions have been refuted by medical literature and reasoning relied upon and employed by the Department of Labor, which is deemed to be a more impartial and reliable evaluative source focused on the public interest rather than the perspective of a private party. Whereas Dr. Fino believes the relationship between chronic obstructive lung disease and coal dust inhalation is not clinically significant, the Department of Labor has found upon review of medical evidence

¹¹ The Department of Labor acknowledges that its position “is consistent with the growing body of case law recognizing that obstructive lung diseases can arise from coal mine dust exposure.” 65 Fed. Reg. 79,943 (December 20, 2000).

and case law, and the amended regulations reflect, that “there is a clear relationship between coal mine dust and COPD and lung dysfunction, and that relationship is likely to be *stronger* than what we are able to measure.” Id. at 79,939.¹² Because Dr. Fino’s opinion in this case categorically excludes the possibility that Claimant’s disabling obstructive lung impairment is due to his occupational exposure to coal dust, and thus is inconsistent with the established premises of the regulations, it is entitled to little weight. 65 Fed. Reg. 79,938 (December 20, 2000)

Of the remaining opinions of record, this tribunal finds the opinion of Dr. Koenig most persuasive, as it is documented, based on objective evidence, well supported by extensive medical literature, some of which provides the premises supporting the amended regulations, and is especially well-reasoned in light of the Claimant’s complex medical condition. Drs. Rasmussen’s and Cohen’s reports are also deserving of substantial weight. Both physicians provided well-reasoned opinions. They documented the objective evidence and explained their conclusions in reference to Claimant’s multiple and medically significant conditions and the medical literature endorsed by NIOSH.

Determining the etiology of Claimant’s disabling respiratory condition is complicated by the effects of his 1981 stroke. All of the physicians agree that a stroke can cause pulmonary impairment that is secondary to respiratory muscle weakness. Therefore, any disability caused by the stroke must be accounted for in order to determine whether or not Claimant’s pneumoconiosis is a “substantially contributing cause” of his totally disabling respiratory or pulmonary impairment.¹³ The Department of Labor, as reflected by the amended regulations, maintains that nonrespiratory or pulmonary disabilities may co-exist with total disability due to pneumoconiosis. 65 Fed. Reg. 79,947.

Dr. Spagnolo did not find that Claimant has a respiratory impairment and concluded that any deterioration in lung function is due to the normal aging process (EX 3). He based his conclusion on the absence of interstitial lung disease and lack of clinical lung disease by physical examination. Through all of his reports, Dr. Spagnolo reiterated his opinion that Claimant’s debilitating condition caused by the stroke prevents him from performing valid spirometry, and, therefore, the only reliable indicator of Claimant’s condition are arterial blood gas studies, which have remained unchanged over the years (EX 10). Dr. Stewart is in agreement with Dr. Spagnolo, as he too believes that Claimant’s disability is entirely attributable to the stroke, and that the disability is muscle weakness as opposed to respiratory impairment

¹² Dr. Koenig also found Dr. Fino’s conclusions flawed and refuted his findings, often citing the same studies relied upon by the Department of Labor in amending the regulations and (CX 3).

¹³ In *Vigna*, the Seventh Circuit held that the miner’s entitlement to benefits was precluded by his disabling stroke because the stroke was unrelated to coal mine employment and occurred before any evidence that the miner was disabled by pneumoconiosis. *Peabody Coal Co. v. Vigna*, 22 F.3d 1388 (7th Cir. 1994). The Department of Labor, and, more recently, the Benefits Review Board, have disagreed with *Vigna*, with the former altering §718.204(c) to emphasize its disagreement with the opinion’s holding, which applies only to the Seventh Circuit. 65 Fed. Reg. 79,946-7; *Oscar Etters v. Peabody Coal Co.*, BRB No. 99-0352 BLA (August 30, 2001).

(EX 4, 9, 18).¹⁴ Drs. Rasmussen, Cohen, and Koenig, however, explained that Claimant's pulmonary function studies have demonstrated an obstructive respiratory deterioration since at least 1992 which cannot be explained by the stroke, because CVAs result in restrictive impairment, and the impairment caused by strokes peaks shortly after the event and then usually improves.¹⁵ As demonstrated by Dr. Cohen, Claimant's pulmonary function studies indicate this very progression (CX 2). Moreover, Drs. Spagnolo's and Stewart's opinions are further refuted by Dr. Koenig, who pointed out that the reproducibility of consistent results belies the assertion that Claimant's CVA weakens him to the point where he cannot provide valid pulmonary function testing results. Therefore, because Drs. Spagnolo's and Stewart's opinions were refuted by Drs. Koenig, Cohen and Rasmussen, their opinions are entitled to little weight.

Most persuasive in Dr. Koenig's opinion is his use of pulmonary function testing to explain the effect of Claimant's stroke on Claimant's total disability, and his calculation of Claimant's loss of lung function due to the stroke. Dr. Koenig noted that, because a stroke exerts its maximum effect (a restrictive one) on lung function shortly after it occurs, and the effect on lung function often improves with time, Claimant's 1981 stroke would not account for his later decline in pulmonary function (CX 3). Incorporating this fact with the objective evidence of Claimant's pulmonary function studies, Dr. Koenig drew the conclusion that "because Mr. Carson's pulmonary function tests were obstructive, not restrictive, because both the DLCO and D/VA were diminished and because a significant decline in lung function occurred many years after Mr. Carson's CVA, a CVA can not explain all of Mr. Carson's pulmonary impairment." *Id.* at 5. Using the Claimant's observed decline in FEV1, Dr. Koenig determined that the stroke resulted in a FEV1 loss of 0.45L (Liters). What this loss demonstrates is that, but for Claimant's 1981 stroke, his FEV1 would have been 0.45L larger, and, even accounting for the 0.45L, i.e., even if Claimant had never had a stroke, Claimant would still be totally and permanently disabled by pulmonary causes (CX-3, 5). In conclusion, Dr. Koenig's opinion provides a reasoned and documented accounting for Claimant's lung function loss attributable to his stroke. Given that none of the other physicians found fault with his analysis, this tribunal finds that Dr. Koenig has convincingly determined that Claimant's totally disabling pulmonary impairment is caused primarily by obstructive lung disease, and not respiratory muscle weakness due to stroke. Accordingly, other physicians' opinions which attribute Claimant's obstructive impairment to his stroke are found less persuasive.

Accounting for the effects of Claimant's stroke allows a more definitive analysis of etiology. Drs. Rasmussen, Cohen and Koenig opined that Claimant's coal dust exposure, in addition to his smoking history, at least significantly contributed to his disabling chronic obstructive impairment. Dr. Rasmussen diagnosed Claimant with complicated pneumoconiosis. While this tribunal finds that diagnosis unpersuasive,

¹⁴ In his final report, Dr. Stewart conceded that if he did accept Claimant's most recent spirometry as valid, there would be evidence of chronic obstructive pulmonary disease attributable to smoking and a decline in lung function since 1992.

¹⁵ Dr. Zaldivar also stated, based on a review of medical literature, that strokes result in restrictive and not obstructive impairment (EX 11).

Dr. Rasmussen's explanation for Claimant's disabling pulmonary impairment is not necessarily erroneous. Dr. Rasmussen opined that Claimant's significant history of coal dust exposure, which he noted primarily occurred prior to the institution of dust suppression in the mines, in conjunction with his cigarette smoking spanning forty-seven years and some effects of his left hemiparesis, account for Claimant's total disability (DX 11). He specifically found that Claimant's coal dust exposure was a significantly contributing factor, relying on medical literature endorsed by NIOSH, indicating that both coal dust and cigarette smoking are known and potent causes of chronic obstructive pulmonary disease (CX 1). Dr. Cohen agrees that Claimant's substantial exposure to coal dust prior to dust control regulations, in addition to his lengthy smoking history, was significantly contributory to the development of his severe obstructive lung disease and severe diffusion impairment (CX 2). Dr. Cohen recognized that pulmonary function studies have consistently shown impairment, even before Claimant's 1981 stroke. Dr. Cohen noted that the 1980 pulmonary function test results indicated a mild restrictive impairment, which progressed to a moderate impairment in the late 1980's, and that there is now significant evidence of a primarily obstructive lung disease. In determining that coal dust exposure contributed to Claimant's disabling obstructive disease, Dr. Cohen relied upon objective evidence of Claimant's radiographically significant pneumoconiosis, his pulmonary function studies, work history, and medical literature describing the effects of coal dust inhalation on smoking miners like Claimant and the progressive nature of pneumoconiosis.

Dr. Koenig concluded that the only sound medical diagnosis for Claimant's disabling chronic obstructive pulmonary disease is that neither coal dust inhalation nor smoking can be excluded as a cause (CX 3). He supported his conclusion with an extensive discussion of the medical literature demonstrating that coal dust exposure, in both smoking and nonsmoking miners, can cause clinically important losses of lung function, and coal dust induced lung disease can progress after the miner leaves the coal mines, even without radiographic and pulmonary function test abnormalities at the time of departure. Dr. Koenig reasoned that Claimant's coal dust exposure cannot be ruled out as a contributing cause of his impairment because, as supported by medical literature, "the symptoms, pulmonary function tests and Chest x-ray appearance of COPD are identical whether caused by coal dust, by cigarette smoke, or by both." (CX 3). Therefore, based on the numerous studies in medical literature and the opinions of NIOSH to which he referred, Dr. Koenig concluded that the only sound medical diagnosis in Claimant's case was not to exclude either smoking or coal dust inhalation as a cause of Claimant's totally disabling pulmonary impairment. He stated that a finding that Claimant's respiratory disability was only due to cigarette smoking would be a total disregard the medical literature, NIOSH, the Industrial Injuries Advisory Council of Great Britain, and numerous experts in the field of occupational lung disease.

This tribunal acknowledges that, at first glance, Dr. Koenig's opinion seems equivocal as to whether coal dust inhalation contributed to Claimant's disabling chronic obstructive pulmonary disease. However, in *Piney Mountain Coal Co. v. Mays*, 176 F.3d 753 (4th Cir. 1999), the Fourth Circuit stated that "a reasoned medical opinion is not rendered a nullity because it acknowledges the limits of reasoned medical opinions." *Id.* at 763. The Court maintained that when a physician uses a conditional term such as "could," which was used by Dr. Koenig, it is proper to consider the use of the term in the context of the physician's entire opinion. Where a complete reading indicates that it is reasonable to read the

conditional terms as “simply acknowledging the uncertainty inherent in medical opinions,” it is improper to reject the opinion as equivocal as a matter of law. *Id.* Upon review of Dr. Koenig’s entire opinion, this tribunal finds that it provides a positive opinion about the etiology of Claimant’s disabling pulmonary impairment, and, rather than being equivocal, simply acknowledges the uncertainty inherent in medical opinions and reaches conclusions with appropriate caution. Dr. Koenig explained his observations, the body of medical knowledge he relies upon, and his own reasoning with certainty; therefore, his opinion is not equivocal or entitled to less weight due to his use of cautious language.

In the judgment of this tribunal, the opinions of Drs. Rasmussen, Cohen and Koenig are entitled to predominant weight. Not only are they consistent with the objective evidence in the case, but they show a persuasive understanding of the medical literature and its interplay with the regulations. It is notable that all of the reports in evidence were written prior to the final issuance of the amended regulations. Many of the studies relied upon by Drs. Rasmussen, Cohen and Koenig provided the premises adopted as underlying the regulations in the official record of rulemaking, and proved to be essential in the incorporation of case law and medicine into the guidelines for awarding benefits under the Act. Moreover, by relying on this medical literature and the opinions of NIOSH, the views expressed in these opinions are both consistent with the regulations and their supporting rationales. The ability to reconcile the objective evidence with the medical literature and to provide documented and well reasoned opinions based on the evidence of record, makes the opinions of Drs. Rasmussen, Cohen Koenig, convincing to this tribunal. It therefore finds that Claimant has established that his pneumoconiosis was at least a substantially contributing cause of Claimant’s respiratory disability, as it has had a material adverse effect on Claimant’s condition.

The reliance of Drs. Zaldivar, Morgan, Castle and Chillag on premises disproved in the medical literature, refuted by the other physicians, and in disagreement with the regulations makes their opinions significantly less convincing. Additionally, the objective evidence of record simply does not support some of their conclusions, especially those reached through inconsistent consideration of Claimant’s condition over the years and in the record of this case, and in light of the standards utilized by the regulations and supported by the medical literature.

Dr. Morgan stated that Claimant’s lung function was “relatively normal” for his race until his stroke in 1981, after which he developed a restrictive impairment and a mild airways obstruction due to his continued smoking. Dr. Morgan stated with certainty that, “the fact remains that prior to his stroke his lung function was adequate and would have in no way impaired his ability to work in the coal mines.” (EX 3). In concluding that Claimant’s coal mine employment and related pneumoconiosis did not contribute to his totally disabling respiratory impairment, Dr. Morgan noted that Claimant’s obstructive impairment did not occur until after he ceased mining but while he continued to smoke. Therefore, Dr. Morgan concluded that the evidence clearly indicates that the cause of Claimant’s impairment is smoking. Dr. Morgan defended his position, stating that he does not disagree that pneumoconiosis can progress after the cessation of coal mine employment, but that he believes that (1) a miner who has simple coal workers’ pneumoconiosis and leaves mining will not show progression of the simple coal workers’ pneumoconiosis and (2) a miner with category two or three simple pneumoconiosis can develop progressive massive fibrosis after he ceases coal

mining (EX 8). Based on these axioms regarding the progression of pneumoconiosis, Dr. Morgan concluded that Claimant's pneumoconiosis did not progress.

While Claimant's lung function may have not been disabling prior to his stroke, the objective evidence of record indicates that as early as 1980, Claimant's lung function was declining. The evidence of record indicates that Claimant's FEV1 was less than seventy percent of predicted in 1980 and continued to decline to less than forty percent of predicted in 1999.¹⁶ Additionally, several physicians have noted that Claimant's pre- and post-stroke pulmonary function testing indicated a mild restrictive impairment, which has dissipated and has been superseded by Claimant's worsening obstructive impairment (See e.g. DX 43-49, 43-26; CX-2). While Claimant's restrictive impairment was not disabling in 1980, it was present. Dr. Morgan reviewed the Claimant's pre- and post-stroke pulmonary function testing and medical evaluations, and, therefore, his determination that Claimant's pulmonary impairment occurred entirely subsequent to his stroke is unpersuasive.

Moreover, Dr. Morgan's opinion regarding the progression of pneumoconiosis after coal dust inhalation has ceased is at odds with the regulations and other well-reasoned opinions of record. As stated earlier, the amended regulations codify the recognition that pneumoconiosis is a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure. §718.201(c). Extensive comments related to amended §718.201(c) explain that the scientific literature strongly supports the latent and progressive nature of pneumoconiosis, and does not differentiate between "simple" and "complicated" pneumoconiosis. 65 Fed. Reg. 79,968-79,973 (December 20, 2000). Moreover, Drs. Rasmussen, Cohen and Koenig all refuted Dr. Morgan's contention, citing numerous studies, also relied upon by the Department of Labor, indicating that coal dust induced lung disease can progress after the miner ceases coal mine employment. Despite Dr. Morgan's lengthy analysis, his opinion is largely inconsistent with the current medical literature cited by the Department of Labor, and it is unpersuasive because deterioration in Claimant's lung function began, albeit mildly, prior to his 1981 stroke. Consequently, Dr. Morgan's opinion is not well-reasoned. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987); *Bogan v. Consolidation Coal Co.*, 6 B.L.R. 1-1000 (1984).

Dr. Zaldivar's opinion is reasoned, but is premised upon an impermissibly restricted conception of legal pneumoconiosis. While his opinion deals with the objective evidence of the case, it is not creditable under the Act and regulations. Though he appears to understand Claimant's physical and physiological condition, Dr. Zaldivar utilizes such a constrained or limited definition of the disease that in this case it effectively precluded a diagnosis of legal pneumoconiosis or attribution of the Claimant's totally disabling pulmonary impairment to legal or clinical pneumoconiosis. Therefore, Dr. Zaldivar's opinion is given little weight

¹⁶ Claimant's FVC values also declined from approximately seventy-one percent of predicted in 1980 to fifty-one percent in 1999. This tribunal notes that the predicted FEV1 and FVC values used for comparison were adjusted to account for Claimant's race. See CX 3.

In his September 28, 1999 report, Dr. Zaldivar diagnosed Claimant with simple coal workers' pneumoconiosis, but found that his respiratory impairment was not caused by pneumoconiosis, but by weakened respiratory muscles (DX 35). In his July 11, 2000 report, based on review of medical records, Dr. Zaldivar recognized "some degree of airway obstruction and emphysema from his ongoing smoking habit," and noted that Claimant had developed more airway obstruction since 1992 (EX 11). However, two months later, during his September 12, 2000 deposition, Dr. Zaldivar stated equivocally that Claimant's decline in FEV1 could be indicative of a true mild obstruction or simply an effect of his respiratory weakness (EX 17 at 19, 31-33, 42). In either case, he discounted pneumoconiosis as a cause of an obstructive impairment in Claimant because he did not observe a radiographic progression in Claimant's x-rays, and because he found that Claimant did not exhibit an obstructive impairment at the time he left the coal mines.

In providing two explicit reasons for ruling out pneumoconiosis as a cause for Claimant's totally disabling obstructive pulmonary impairment, Dr. Zaldivar disclosed his basis for Claimant's entire diagnosis. During the September 2000 deposition, Dr. Zaldivar enunciated the two reasons as follows:

Q: What about the possibility that some of this increased obstructive impairment is the result of his coal workers' pneumoconiosis?

A: No, that's not likely. There is no progression radiographically. And the amount of damage to the lungs is caused by the amount of dust and the reaction to the dust in the lungs. His chest x-ray has remained stable, so there is no reason to think that there is anymore reaction to the lungs [or] in the lungs now to the dust than there was in 1989 or earlier because of progression of obstruction.

Q: Can coal workers' pneumoconiosis in fact progress?

A: Well, that was actually answered in both radiographical terms and breathing test terms. If there is airway obstruction present at the time the individual ceases the mine work and there is - - and this obstruction is due to mine work, then the obstruction will progress faster simply because the individual ages and, as we age, we lose lung capacity. (EX 17 at 33-34).

Q: Did you agree or disagree with his [Dr. Rasmussen's] opinion that both cigarette smoking and coal mine dust exposure should be considered contributing factors to the disabling respiratory insufficiency?

A: Well, I disagree with it for the reason I have mentioned. It is true that he has continued to smoke, Mr. Carson has continued to smoke. And smoking does cause airway obstruction. And if he has any airway obstruction now, it is the result of his smoking habit because it wasn't there when he quit work in the mines even before the stroke. The FEV1 percent was entirely normal. And the profusion and

the chest x-ray haven't changed as I explained earlier. And that simply means that there is no more reaction to the dust in the lungs than there was all along even before the stroke.

So, coal mining didn't cause any obstruction back before the stroke, and it isn't causing any obstruction now either. It is the smoking that's causing obstruction, if in fact obstruction is present. (EX 17 at 42).

Q: A miner's impairment can progress even in the absence of radiographic progression?

A: Only if there is airway obstruction present at the time they cease to work, and I already mentioned that as well during the early part of this deposition in detail. (EX 17 at 56-57).

Thus, Dr. Zaldivar sets out his restricted diagnostic definition of pneumoconiosis. Through his use of evidence and his express reasoning, Dr. Zaldivar reveals that, while his understanding of clinical pneumoconiosis is consistent with that of the Act, his misconception of legal pneumoconiosis is so restrictive, that it contravenes the Act and regulations and, by its application, effectively precludes a finding by the doctor of any circumstances of totally disabling legal pneumoconiosis in this Claimant.

It is evident from Dr. Zaldivar's deposition testimony that he in essence recognizes clinical pneumoconiosis as a radiographically visible retention of coal dust in the lungs. He diagnosed Claimant with simple coal workers' pneumoconiosis based on a finding of such radiographic evidence (DX 35; EX 11, 17 at 12-13). However, he did not find that Claimant's clinical pneumoconiosis contributed to any disabling pulmonary obstructive impairment that Claimant may have because he opined that progression of the disease into a disabling pneumoconiosis would not occur in the absence of radiographic progression of coal macule size and/or profusion. Dr. Zaldivar testified that he did not personally observe such progression in Claimant's case. Thus, while Dr. Zaldivar's opinion that clinical pneumoconiosis did not cause any obstructive impairment in this Claimant may be reasoned, it does not account for the progression or effects of any legal pneumoconiosis or its relation to Claimant's disabling pulmonary impairment, which is clearly established and recognized by Drs. Koenig, Cohen and Rasmussen.

Applicable regulations and case law recognize that both clinical and legal pneumoconiosis can exist in the absence of radiographic evidence. §718.202(b); *see generally Nagle v. Barnes & Tucker Co.*, 1 B.L.R. 1-961 (1978). The "legal" definition of pneumoconiosis is much broader than the clinical definition, which encompasses lung diseases characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by coal dust exposure in coal mine employment. §718.201(a)(1). Because the forms of pneumoconiosis under the umbrella of the legal definition under the Act can be present and disabling without producing the trademark fibrotic reaction of clinical pneumoconiosis, radiographic evidence cannot be the exclusive determinant of whether legal pneumoconiosis has progressed or worsened, as Dr. Zaldivar suggests. By noting that

Claimant's x-rays have not shown a progression of increased size or profusion of visible macules, Dr. Zaldivar demonstrates only that Claimant's clinical pneumoconiosis has not progressively worsened over time.¹⁷ His demonstration obviously could not reveal any progression of Claimant's legal pneumoconiosis. Indeed, under the constraints imposed by his restructured definition, Dr. Zaldivar ruled out both coal mine employment as a cause of Claimant's totally disabling pulmonary impairment and the possibility that Claimant had legal pneumoconiosis in several reports and depositions which comprise the entirety of Dr. Zaldivar's opinion.

As thus revealed, Dr. Zaldivar stated that when pneumoconiosis is responsible for a pulmonary impairment, it causes a purely obstructive defect. In 1989 and 1992, Dr. Zaldivar, based on examination of the Claimant and review of medical reports, stated that Claimant had a mild restrictive impairment prior to his stroke, and opined in this record that pre-stroke pulmonary function testing had indicated reduced FEV1 and FVC (DX 43-26). He opined that the restriction was unrelated to Claimant's pneumoconiosis or coal mining employment, stating:

"These studies show that Mr. Carson had a mild restriction of his vital capacity. There was absolutely no evidence of obstruction. There are many causes of restriction, but none of them are coal workers' pneumoconiosis. Coal workers' pneumoconiosis, when it causes any sort of impairment, does so by causing an airway obstruction." (DX 43-26).¹⁸

The legal definition of pneumoconiosis, however, is broader than the definition Dr. Zaldivar

¹⁷ Dr. Zaldivar considered his own readings of films from 1989 and 1999 in determining that no radiographic progression occurred. (EX 17).

¹⁸ Dr. Zaldivar's categorical opinion stands in striking contrast with the medical opinions approved in *Stiltner*, where the Fourth Circuit observed, "Unlike the medical opinions we examined in *Warth*, none of the challenged physicians here assumed that coal mine employment can never cause COPD; they merely opined that Stiltner likely would have exhibited restrictive impairment in addition to COPD, if coal dust exposure were a factor." (Footnotes omitted.) *Stiltner v. Island Creek Coal Co.*, 86 F.3d 337, 341, 20 B.L.R. 2-246, 2-254 (4th Cir. 1996). Apparently Dr. Zaldivar once shared the view of the physicians approved in *Stiltner*, because the Fourth Circuit noted in *Stover v. Consolidation Coal Co.*, 45 F.3d 427 (4th Cir. 1995):

The ALJ accorded "full weight" to the opinions of Drs. Zaldivar, Fino, and Loudon, who found no pneumoconiosis, based on their expert credentials, and those physicians found that the miner's disabling respiratory impairment was solely attributable to the miner's extensive smoking history and lifelong asthma. They explained how the miner's pulmonary function studies reflected the presence of an obstructive form of respiratory impairment rather than the restrictive form associated with pneumoconiosis."

This observation by the court tends to impair further the credibility of Dr. Zaldivar's inconsistent opinion in this case.

obviously applied, and encompasses “any chronic lung disease or impairment and its sequelae arising out of coal mine employment.” §718.201(a)(2). It includes, but is not limited to, “any chronic **restrictive or obstructive** pulmonary disease arising out of coal mine employment.” *Id.* (emphasis added). By limiting his diagnosis of pneumoconiosis to obstructive pulmonary disease only, Dr. Zaldivar failed to include consideration of legal pneumoconiosis within the scope of that diagnosis, and in doing so, precluded any possible diagnosis of Claimant’s restrictive pulmonary disease as pneumoconiosis which could be plausible under the Act. However, Dr. Zaldivar does not stop there in his distortion of the regulatory definition.

Dr. Zaldivar stated that even if a patient has an obstructive impairment, it can only be linked to the patient’s occupational exposure if the impairment was present at the time the patient left his coal mine employment. In his deposition of April 13, 1992, the following exchange took place:

Q: Do you expect impairment due to pneumoconiosis to progress absent further dust exposure?

A: No. If there is no impairment found at the time the individual ceases to work, one does not find any pulmonary impairment subsequent to that. (EX 43-41 at 28).

During his subsequent September 12, 2000 deposition, Dr. Zaldivar affirmed his opinion and reiterated his belief that pneumoconiosis causes a purely obstructive impairment. He stated that a miner’s impairment can only progress in the absence of radiographic progression “if there is airway obstruction present at the time they cease work.” (EX 17 at 57). Therefore, for the past decade, Dr. Zaldivar has repeatedly affirmed his opinion in the context of this claim that pneumoconiosis is not a latent disease, and that if it does progress, it will only do so if it was present at the time the exposure to coal dust ended.

Recent amendments to the regulations codified case law to explicitly include within the definition of pneumoconiosis, the following refinement:

For purposes of this definition, “pneumoconiosis” is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure. §718.201(c).

Therefore, by not recognizing pneumoconiosis as a latent and progressive disease process, Dr. Zaldivar has relied upon a definition that is both overly restrictive and is in conflict with the regulations.

Finally, while Dr. Zaldivar has established that he will only diagnose pneumoconiosis based on a finding of an obstructive impairment at the time a patient leaves the coal mines, he has declared that he does not expect to find such an impairment. At the September 12, 2000 deposition, the following exchange took place between Claimant’s attorney and Dr. Zaldivar:

Q: Okay. The inhalation of coal mine dust can cause obstruction?

A: Inhalation of coal mine dust can cause obstruction, yes.

Q: We know that coal mine dust can cause obstruction because of epidemiological studies?

A: Well, we know that coal dust can cause a drop in FEV1 in a certain group of coal miners because of epidemiological studies, but we don't have any animal data to show that as we have in the smoking data. There's a difference.

Q: Both cross sectional and longitudinal studies have shown the relationship between coal mine dust exposure and the development of obstructive lung disease?

A: Not as an invariable result. It is - - It is shown that individuals do develop airway obstruction, but there is not a - -. Either result is not [an] expected outcome of mining. The minority of miners develop obstruction.

Q: What percentage would you say develop obstruction?

A: Well, I do know from the data from the Occupation and Labor Board, my understanding is that at least less than four percent develop significant obstruction, ten percent develop radiographic pneumoconiosis, and somewhere in between the ten and five percent are individuals who develop some degree of pulmonary impairment. One percent of those individuals who develop simple pneumoconiosis may develop complicated pneumoconiosis.

So, these account information available. We're not talking - - We're not speaking about the majority of coal miners. (EX 17 at 76-77).

Dr. Zaldivar's testimony indicates a conviction that only a very small minority of miners will develop an obstructive impairment due to coal mine employment, and that an even smaller minority will develop a significant impairment, which in turn suggests that he does not expect to witness the pulmonary obstruction he claims is indicative of coal dust induced pulmonary impairment. Again, this expectation further narrows the definition of pneumoconiosis contemplated by Dr. Zaldivar.

Dr. Zaldivar's three stated premises reduce the operative definition of legal pneumoconiosis for Dr. Zaldivar to, "Pneumoconiosis is an obstructive pulmonary impairment that is not latent and rarely progressive, and is not an expected outcome of coal mine employment." Such a definition is far narrower than that contemplated by the Act and regulations. Consequently, Dr. Zaldivar's opinions based on his effectively restricted definition are fundamentally at odds with the Act. Furthermore, because Claimant's obstructive impairment was not significantly present at the time he left the coal mines, Dr. Zaldivar was

methodically preempted, within his own self-imposed constraints, from diagnosing Claimant with legal pneumoconiosis. Nor could he attribute Claimant's obstructive pulmonary impairment to his coal dust exposure. Consequently, his opinion as to causation, though reasoned, cannot be accorded substantial weight.

The remaining opinions of record are those of Drs. Chillag and Castle. Dr. Chillag found evidence of simple coal workers' pneumoconiosis and a pulmonary impairment "probably" related to Claimant's stroke and possibly his cigarette smoking (DX 39). He found the Claimant totally disabled due to his stroke and and vascular disease and related the pulmonary impairment to Claimant's muscle weakness and smoking (DX 39; EX 7). Dr. Chillag did not opine that the disability is related to Claimant's pneumoconiosis. Dr. Chillag did not provide reasoning until his supplemental report where he explained that Claimant's disability occurred due to his stroke, which was prior to the demonstration of significant pulmonary impairment (EX 15). Dr. Chillag further stated that Claimant's general condition has deteriorated due to his advanced age. *Id.* Dr. Chillag's opinion provides little analysis and is not documented. As with Drs. Zaldivar and Fino, he did not explain why Claimant's decreasing pulmonary function was not deemed significant until it became significant in regard to disability. Moreover, in determining that Claimant's age and general medical conditions are responsible for his continued deterioration, Dr. Chillag did not address the fact that at that age, Claimant had experienced over thirty years of coal mine employment, nor did he discuss how he ruled out any contribution from such employment. There is simply no indication that he even considered Claimant's employment history. *See Pinansky v. Director, OWCP*, 7 B.L.R. 1-171 (1984). Therefore, this tribunal accords Dr. Chillag's opinion very little weight.

In three reports and a deposition of record, Dr. Castle diagnosed Claimant with simple coal workers' pneumoconiosis and mild obstructive lung disease attributable to Claimant's ongoing tobacco habit (EX 4). He explained that the obstruction is not attributable to Claimant's pneumoconiosis because it only became apparent eighteen years after Claimant left the mines, and because, "When coal workers' pneumoconiosis causes impairment it does so by causing a mixed, irreversible obstructive and restrictive ventilatory impairment," and Claimant does not have a restrictive impairment.¹⁹ *Id.* Dr. Castle found Claimant totally disabled due to his previous stroke and peripheral vascular disease, both of which he concluded are severe. He also eliminated Claimant's pneumoconiosis as a cause of his continuous decline in lung function, citing the lack of radiographic evidence of progression (EX 14).

Dr. Castle's opinion is unpersuasive regarding the etiology of Claimant's pulmonary impairment for the same reason as Dr. Zaldivar's. Although Dr. Castle discusses the difference between legal and clinical pneumoconiosis, his opinion fails to distinguish the etiology of the clinical pneumoconiosis from that of the more broadly defined legal pneumoconiosis. Such reasoning does not provide an opinion regarding

¹⁹ During his deposition, Dr. Castle stated that the mixed impairment is not a generality, stating that "nothing is 100 percent in medicine." (EX 16).

the etiology of legal pneumoconiosis, and, therefore, is not probative with respect to that issue (EX 16). *Shonborn v. Director, OWCP*, 8 B.L.R. 1-434, 1-436 (1986). Like Dr. Zaldivar, Dr. Castle maintains that one cannot determine a miner's impairment by looking at his x-rays, and that x-rays may underestimate the amount of pneumoconiosis. He does not entirely disagree that pneumoconiosis can be disabling in a miner who has a normal x-ray, but he has never seen this occur. Moreover, he would not answer Claimant's question on cross-examination, whether he required an x-ray to find total disability due to pneumoconiosis. Like Dr. Zaldivar, Dr. Castle, it appears, only considers clinical pneumoconiosis in forming his opinions. Therefore, his opinions are not probative of whether Claimant's total disability is due to pneumoconiosis, and this tribunal accords his numerous opinions little weight. *Id.*

This tribunal has reviewed the opinions of the ten similarly qualified physicians. In resolving this battle of the experts through evaluation of their divergent opinions, this tribunal carried out its province under the APA²⁰ to initially determine whether each medical report of record relevant to the issue was reasoned and documented, and it has provided reasons for discounting opinions. *See Collinks v. J & L Steel*, 21 B.L.R. 1-181 (1999). In making these determinations, this tribunal considered the physicians' qualifications, their reasoning, their reliance on objectively determinable symptoms and established science, their detail of analysis, and their freedom from irrelevant distractions and prejudices. *Underwood v. Elkay Mining, Inc.*, 105 F.3d 946, 951 (4th Cir. 1997); *see also Milburn Colliery Co. v. Hicks*, 138 F.3d 524 (4th Cir. 1998). Additionally, this tribunal paid close attention to the spirit, underlying rationales, and supporting case law and scientific evidence of the regulations. The opinions of those physicians finding that Claimant's pneumoconiosis at least substantially contributed to his totally disabling pulmonary impairment provided the most persuasive reasoning in light of the many factors considered. These physicians' opinions accounted for every aspect of Claimant's physical condition before and after his 1981 stroke, documented and explained their reasoning, and supported their conclusions with science explicitly subsumed in the applicable regulations. While this tribunal acknowledges that there may remain some areas of debate, the opinions of seven physicians were unpersuasive in light of the factors considered. In general, those physicians did not account for Claimant's observable pre-stroke pulmonary impairment, were not in accord with the accepted body of medical literature subsumed in the regulations, did not distinguish in their reasoning the etiologic differences between clinical and legal pneumoconiosis, and seemed unwilling to consider Claimant's extensive coal mining history in relation to his smoking history.²¹ Therefore, in consideration of the above reasoning, this tribunal finds that, based on the more persuasive opinions of Drs. Koenig, Rasmussen and Cohen, Claimant has established that his pneumoconiosis substantially contributed to his totally disabling pulmonary impairment, and has proved a material change in conditions.

²⁰ 5 U.S.C. §557(c)(3)(A), as incorporated into the Black Lung Act by 5 U.S.C. §554(c)(2), 33 U.S.C. §919(d), 30 U.S.C. §932(a).

²¹ This tribunal notes that it was the former three physicians, who found contribution from Claimant's pneumoconiosis, who utilized the most extensive smoking history reported (approximately forty-seven years in length) in reaching their conclusions.

Entitlement

In conclusion, because Claimant has established total disability due to pneumoconiosis. He has established a material change in conditions, and, accordingly, entitlement to black lung benefits.

Date of Onset

The Claimant was physically disabled by unrelated causes before his pneumoconiosis had gradually progressed enough to contribute substantially to his disability. The last denial in 1992, affirmed in 1996, recognized total disability that was not caused by coal workers' pneumoconiosis. Neither the medical tests nor the physicians' opinions pinpoint when pneumoconiosis could be considered to be a substantial cause of the Claimant's disability. The date of onset, therefore, cannot be deduced from the evidence of record, and so payment of benefits upon proof of entitlement should commence as of November 1998, the month in which the instant claim was filed. §725.503(b).

Attorney's Fees

An attorney's fee is approvable in accordance with 20 C.F.R. §§725.366 and 725.367. Application for such approval, with service upon Claimant and all other parties, should be filed within thirty (30) days of the date of this decision. Parties may file objections within ten (10) days following receipt of such an application. The Act prohibits charging a fee without prior approval pursuant to these applicable regulations.

ORDER

The claim of William H. Carson for black lung benefits under the Act is hereby granted. Respondent Westmoreland Coal Company is directed to pay to Claimant all black lung benefits to which he is entitled, augmented with respect to his two dependents, commencing as of November 1, 1998.

A
EDWARD TERHUNE MILLER
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this notice must also be served on Donald S. Shire, Associate Solicitor, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.